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PESTS NOT KNOWN TO OCCUR IN THE UNITED STATES OR OF LIMITED DISTRIBUTION NO. 88: PLUM POX VIRUS

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Disease

Plum pox, pox, sharka, or sarka of apricot, peach, Japanese plum, or plum

Pathogen

Plum pox virus (PPV) Atanasoff 1932

Other Names

Prunus virus 7 Christoff 1938

Group

Potyvirus. Cryptogram R/1:3.5/6.2±0.4:E/E:S/Ap (Nemeth 1986)

Economic Importance

Plum pox virus causes serious losses to plums, apricots, peaches, and Japanese plums in Europe. Fruit loss can be total in susceptible cultivars. PPV reduces the number of fruit by affecting pollination and increasing fruit drop. Average plum yield per tree dropped about 83 percent from 1949 to 1963 in a heavily infested district of Czechoslovakia. The fruit was unsalable (Blattny and Heger 1965). External and internal appearance, flavor, and shelflife decline (Pemberton 1980, Zawadzka and Millikan 1971).

As there is no cure or treatment, infected trees are usually removed. The costs of replacing diseased trees with healthy ones, of production loss due to replacement, and of intensive inspection and survey must also be considered part of the economic losses. Tree replacement in one orchard in Poland for a very susceptible plum cultivar totaled about 18 percent in 15 years (Grzyb 1984). Replacement is much higher in the Balkans, where the disease is more severe (P. R. Fridlund, pers. comm.).

Hosts

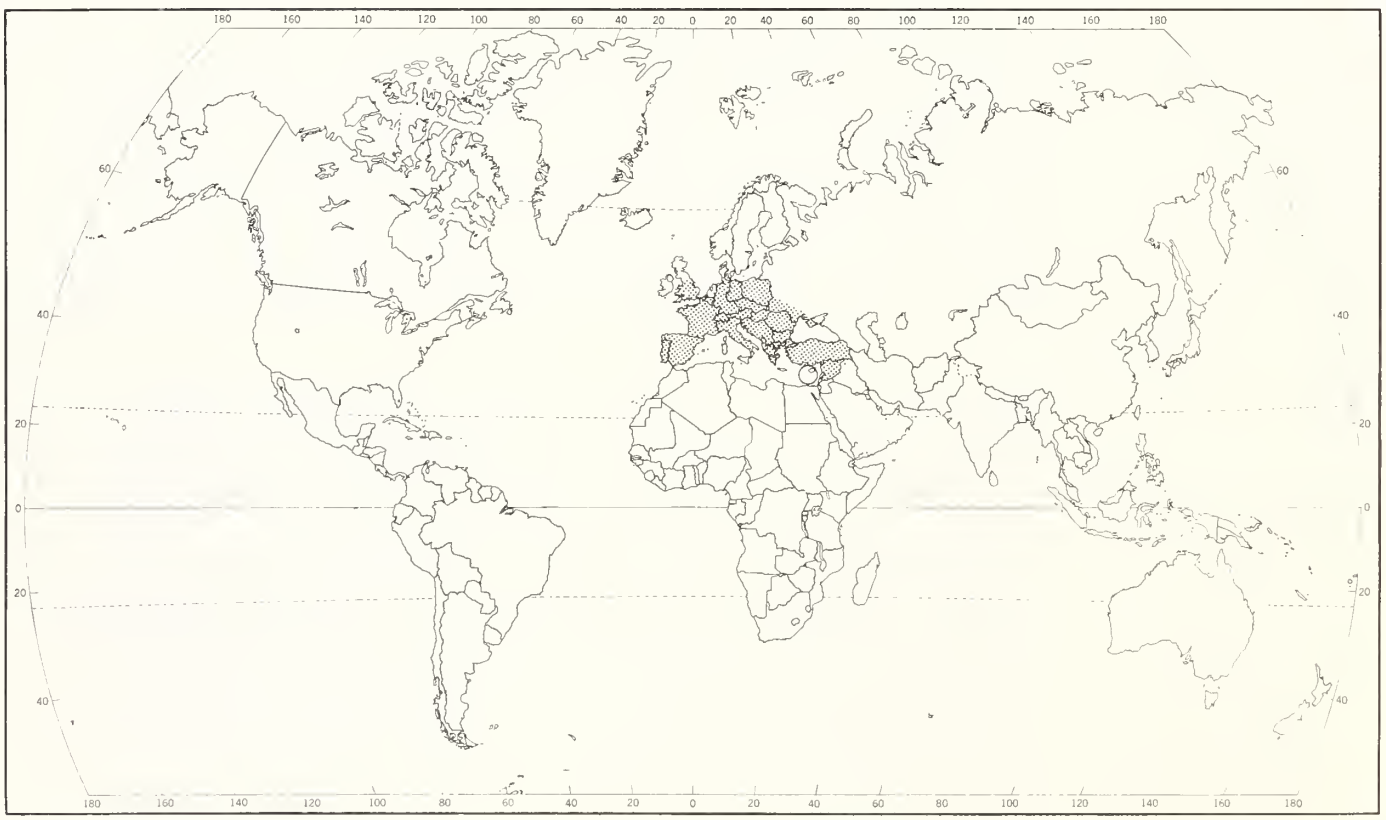
Woody hosts appear to be restricted to the genus *Prunus*. Species reported as naturally infected include *P. armeniaca* (= *P. divaricata* (European and Mediterranean Plant Protection Organization 1970), apricot (Mathys 1974)); *P. cerasifera*, myrobalan plum (European and Mediterranean Plant Protection Organization 1970, Mathys 1974); *P. domestica*, plum (Mathys 1974), *P. domestica* subsp. *insititia*, damson plum (Trifonov 1978); *P. dulcis*, almond (Llacer et al. 1985b); *P. glandulosa* (Nemeth and Schmelzer 1972); *P. persica*, peach (Mathys 1974); *P. salicina* (= *P. triflora*), Japanese plum (Llacer et al. 1985b); *P. spinosa*, blackthorn, a symptomless carrier (Jordovic et al. 1971); and *P. tomentosa* (European and Mediterranean Plant Protection Organization 1970).

Infected herbaceous plants may serve as a potential inoculum source, but no natural transmission of PPV from herbaceous to *Prunus* hosts has been demonstrated although experimental

transmission has occurred. Herbaceous species naturally infected in orchards include: Campanula rapunculoides, creeping bellflower (Kroll 1973); Chenopodium sp. (Llacer et al. 1985b); Lamium album, white deadnettle; Lupinus albus, white lupine; Lycium barbarum (= L. halimifolium), Barbary matrimonyvine (Kroll 1973); Dimorphotheca aurantiaca hybrida (Zawadzka and Smolarz 1978); Medicago lupulina, black medic; Melilotus officinalis, yellow sweetclover (Kroll 1973); Pisum sativum, garden pea; Ranunculus acer (Zawadzka and Smolarz 1978); Silene vulgaris, bladder campion (Kroll 1975); Solanum dulcamara, bittersweet nightshade; Trifolium incarnatum, crimsom clover (Kroll 1973); T. pratense, red clover (Zawadzka and Smolarz 1978); T. repens, white clover (Kroll 1973); and Zinnia violacea (= Z. elegans), zinnia (Zawadzka and Smolarz 1978).

General Distribution

Unless otherwise cited, the European and Mediterranean Plant Protection Organization (1983) and the Commonwealth Mycological Institute (1970) listed the following countries: Albania, Austria, Belgium (local infestation under eradication--van



Plum pox virus distribution map.

Melckebeke pers. comm. 4/15/87), Bulgaria, Cyprus (detected in 1982; eradication underway--European and Mediterranean Plant Protection Organization 1984), Czechoslovakia, Denmark (only Fyn Island, eradication underway--European and Mediterranean Plant Protection Organization 1986), East Germany, France (Commonwealth Mycological Institute 1980), Greece, Hungary, Italy, Poland, Portugal (local infestation under eradication--European and Mediterranean Plant Protection Organization 1985), Romania, Soviet Union (Crimea, Moldavia, West Ukraine--Pomazkov and Abramova 1971), Spain (Llacer et al. 1985b), Switzerland, Syria (Food and Agriculture Organization of the United Nations 1986), Turkey (Sahtiyanci 1969), United Kingdom (England and Wales--Pemberton 1980, D. L. Ebbels, pers. comm. 3/31/87), West Germany, and Yugoslavia.

Characters

Virus particles filamentous (Fig. 1), about 725-760 nm long by 20 nm wide. Three strains exist: necrotic, intermediate, and yellow (Sutic et al. 1971). Needlelike and pinwheel inclusions in nucleus and cytoplasm of leaves and ripe fruit of peach and plum. Inclusions absent from sharka-free and from pseudopox fruit (Kegler and Schade 1971, van Oosten 1971, 1972).

Transmission - Through grafts, sap inoculation (Kegler and Schade 1971) and Prunus seed. Seed transmission rates ranged up to 13.9 percent (Coman and Cociu 1976 from Nemeth 1986; Mathys 1974, Nemeth and Kolber 1983). Coman and Cociu (1976) reported 20-80 percent transmission for pollen.

(Fig. 1)

PPV particles from peach leaf cells after differential centrifugation and negative staining with phosphotungstic acid (X 33,600)(From Maroquin and Rassel 1976).

Several aphid species transmit PPV in a nonpersistent, styletborne manner (Kassanis and Sutic 1965; Kunze and Krczal 1971). No transmission to aphid progeny has been reported. All of the following vectors except four species were found by Kunze and Krczal (1971) and Krczal and Kunze (1972): Aphis citricola Van der Goot (= A. spiraecola Patch), spirea aphid (Leclant 1973); A. craccivora Koch, cowpea aphid (Leclant 1973); Brachycaudus cardui (L.), thistle aphid; B. helichrysi (Kaltenbach), leafcurl plum aphid; Hyalopterus pruni (Geoffroy), mealy plum aphid (Minoiu 1973); Myzus persicae (Sulzer), green peach aphid; M. varians Davidson (Leclant 1973); and Phorodon humuli (Schrank), hop aphid.

After 3 hours of starvation, the aphid acquires PPV (Kunze and Krczal 1971, Krczal and Kunze 1972) in 1-5 minutes of feeding (Kassanis and Sutic 1965, Leclant 1973, van Oosten 1970). PPV persists for 1 hour in M. persicae and P. humuli, but persists for 3 hours in P. humuli if the aphid is starved for 3 hours before and after acquisition (Krczal and Kunze 1972).

Testing - PPV is difficult to detect due to uneven distribution and low concentration of virus in the host. Enzyme-linked immunosorbent assay (ELISA) can detect low PPV concentrations in roots, bark, fruit, seed, flowers, and leaves. Because results are more reliable after, rather than before, foliar symptoms appear (Hamdorf 1983) and because of uneven virus distribution, ELISA is limited to confirming the presence, but not the absence, of PPV (Adams 1978a, Clark et al. 1976, Nemeth and Kolber 1983, Roggero and Lenzi 1985, Torrance 1981).

Slower tests include transmission to sensitive indicator plants (indexing). Grafting infected buds on seedlings of woody indicators (P. persica and P. tomentosa) in early spring results in symptoms in 1-8 weeks (European and Mediterranean Plant Protection Organization 1983, Nemeth 1986). The latter species served as a reliable woody indicator for 18 PPV isolates and separated PPV from 3 other viruses of plum (Rankovic 1975, 1980). Sap inoculation to the herbaceous indicator Chenopodium foetidum reliably produces symptoms only in spring in 6-8 days (European and Mediterranean Plant Protection Organization 1983).

Characteristic Damage

Infected leaves may show chlorotic flecks, bands, rings, and mottling or vein clearing. Infected fruits are discolored and drop prematurely. Symptoms vary with the locality, season, Prunus species, and cultivar. Some cultivars are symptomless.

(Fig. 2-3)

2

3

PPV symptoms on 'Canino' apricot. 2. Leaves. 3. Fruit (Courtesy J. Foster).

(Apricot)

Apricot leaves develop diffuse pale green rings and lines (Fig. 2). Fruit are deformed with chlorotic yellow rings on the skin (Fig. 3). Yellow rings and spots on the stones (Dunez 1987) separate plum pox from other apricot pox (Mathys 1974). Affected trees may be sensitive to drought and cold (Christoff 1958).

(Figs. 4-5)

4

5

Symptoms on 'Red Beaut' Japanese plum. 4. Leaves (far left leaf is healthy). 5. Fruit (From Llacer et al. 1985a).

(Japanese plum)

Japanese plum leaves may show chlorotic rings, bands, and mottling (Fig. 4), later becoming necrotic. Fruit (Fig. 5) show rings, flecks, depressed brownish areas, and conspicuous malformations at ripening (Llacer et al. 1985b).

(Figs. 6-7)

6

7

Symptoms on peach. 6. Leaves of 'GF 305'. 7. Fruit of 'Rojo del Rito' (6, courtesy J. Foster; 7 from Llacer et al. 1985a).

(Peach)

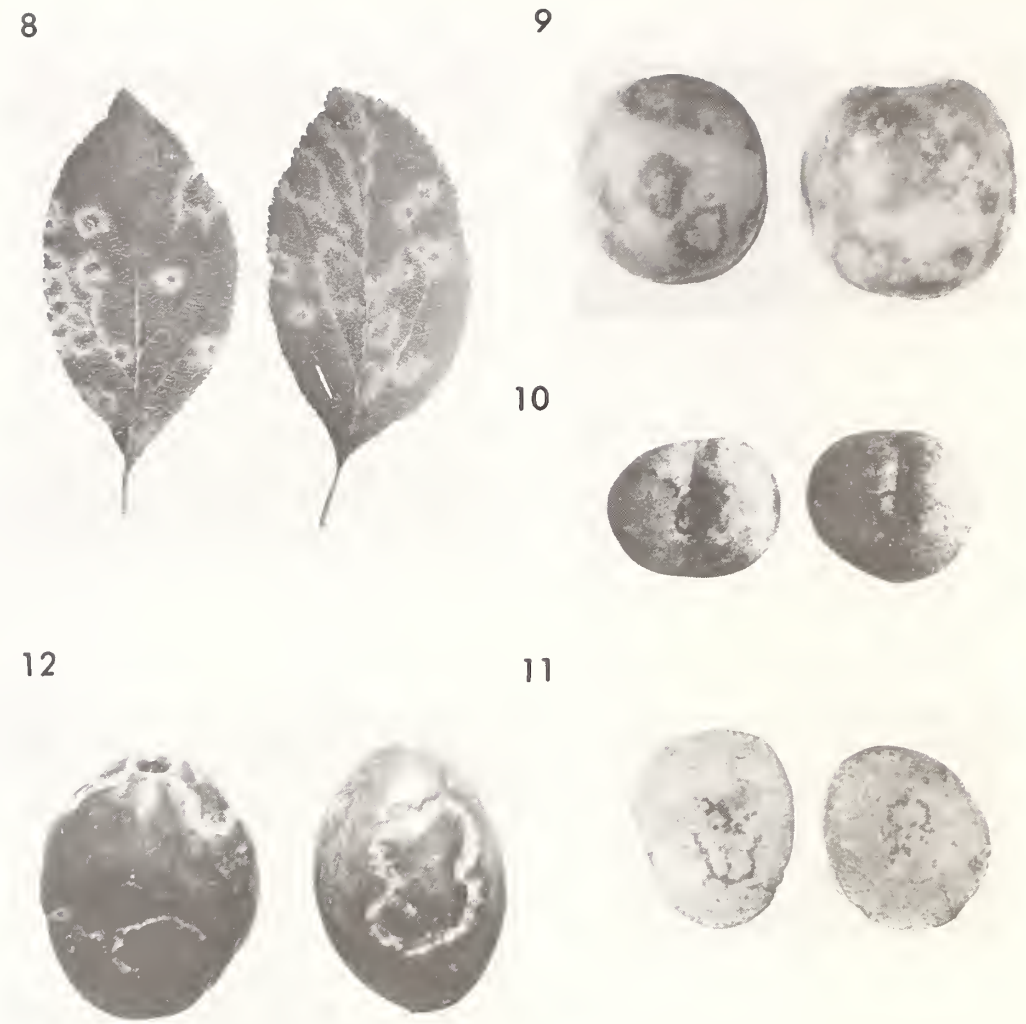
Peach leaves on young shoots show secondary and tertiary vein clearing in early spring. Leaves may be malformed (Fig. 6) with a necrotic main vein and may show some epinasty. Peach fruit develop spots and rings (Fig. 7): white or green on white-fleshed cultivars and greenish yellow to green on yellow-fleshed cultivars. Symptoms also disappear quickly (European and Mediterranean Plant Protection Organization 1983).

(Plum)

Plum leaves may show irregular vein clearings or a dark oak leaf pattern bordering the main and secondary veins. Later, characteristic yellowish to olive green flecks, bands, rings, and mottling appear (Fig. 8). Ring edges are typically distinct on the inner edge and blurred on the outer. Symptoms fade during hot months except in some cultivars where the rings and spots later develop reddish brown margins (Nemeth 1986, European and Mediterranean Plant Protection Organization 1983).

Plum fruit develop red flecks and broad rings on light-skinned fruit; flecks disappear after a short time on dark-skinned cultivars (European and Mediterranean Plant Protection

(Figs. 8-12)



Symptoms on plum. 8. Leaves. 9-12. Fruit. 9. Bands on 'Fleuriana'; 10. Thin, red rings on 'Early Laxton'; 11. Thin, red lines on 'Early Laxton'; 12. Grooves and pits (8 and 12, courtesy B. Zawadzka, Research Institute of Pomology and Floriculture, Skierniewice, Poland; 9-11 from van Oosten 1971b).

Organization 1983). Some cultivars with fruit that are orange, red, or purple (not green or yellow) show broad red/purple bands--diffuse on one side, distinct on the other side (Fig. 9) with thin red rings (Fig. 10) and lines (Fig. 11) near the fruit apex 2-4 weeks before picking. These symptoms are most apparent before the color darkens (van Oosten 1971, 1972).

Plum fruit may show bluish grooving and pitting (Fig. 11) with brownish-red necrosis of the underlying tissue and internal gummosis to the stone. Rings and spots may occur on the stone. Depending on the cultivar, fruit may drop prematurely (Nemeth 1986). In some cultivars, these symptoms are not always an indication of plum pox (van Oosten 1972).

To help distinguish similar symptoms caused by PPV and other viruses such as apple chlorotic leafspot virus (incitant of pseudopox), plum line pattern virus, Prunus necrotic ringspot virus, and prune dwarf virus, see tables in Kegler and Schade (1971) and Mathys (1974).

Detection Notes

Movement of infected propagative material can introduce PPV into new areas. Besides infected vegetative material (plants, roots, scions, and budwood), infected seeds of apricot, peach, and plum may pose a risk in that the resulting young trees may become inoculum sources. Domestic aphids may acquire and spread the virus from imported Prunus cut flowers to nearby fruit trees or weeds. Or, the virus may enter this country via aphid vectors hitchhiking on infected cut flowers. Infected weed hosts would be a problem if the plants were moved about.

Several problems impede early detection of infected material, increasing the probability of introduction and establishment. Roots or other parts may be infected, but the tree appears symptomless. Several years may lapse before symptoms become obvious. Careful inspection is needed to spot the earliest symptoms on newly infected trees before secondary spread occurs to uninfected trees. Symptoms may be difficult to spot because the virus spreads incompletely in the tree and may remain in a small part, for example, a twig for some time. Symptoms may be indistinct and disappear in high summer temperatures. Fruit symptoms may be apparent for less than a month before harvest (Adams 1978b, Pemberton 1980).

To minimize the risk of introducing PPV, propagative material and cut flowers of its primary Prunus hosts is prohibited or restricted entry into the United States by Title 7, Part 319.37 of the Code of Federal Regulations. Propagative material is enterable under USDA permit for scientific purposes subject to stringent entry conditions including testing for PPV.

Survey--

1. Between late spring and early summer and in fall, check young trees for leaves with chlorosis or vein clearing at least twice a season, and inspect infested orchards at least once every 3 weeks. Inspect fruiting and mother trees (for propagation of budwood) several times a year (Pemberton 1980).

2. Watch for heavy, premature fruit drop. Examine fruit during the month before harvest; also inspect dropped or harvested fruit. Look for lines, rings or diffuse mottling on fruit and rings and spots on stones.

Submit for diagnosis, suspect plant material labeled and packaged to preserve its freshness as long as possible.

Biology and Etiology

Infected Prunus trees provide the major source of PPV inoculum. From infected Prunus material, the virus is spread through grafts and budding material during propagation, or by its aphid vectors.

The number of infected trees corresponds to the number of winged vectors present on host fruit trees during the year. Beginning in late winter or early spring, overwintered aphid eggs hatch on PPV-infected fruit trees. After a few wingless generations, winged forms appear, probe or feed on the fruit tree host, and fly to other hosts to also probe and feed. In midseason, the aphids migrate to herbaceous hosts. When these plants slow their growth or die, or when the season cools in late summer, the winged generation develops and flies to stone fruit trees to lay overwintering eggs. These adults move frequently from tree to tree, probing and feeding for short periods (Kunze and Krczal 1971, Pemberton 1980, Pomazkov and Abramova 1971).

Long-distance virus dissemination by the vector may be possible. As mentioned earlier, P. humuli can transmit the virus 3 hours after acquisition if starved before and after feeding. Migrants fast for such periods in traveling to and from a distant infection source (Krczal and Kunze 1972).

Once a tree is inoculated, incubation may take 9-13 months. Systemic spread is slow and may take another 2-3 years in a young tree (Christoff 1958, European and Mediterranean Plant Protection Organization 1983).

Diseased trees first appear in a previously uninfested orchard in a random pattern. After 2 years, however, newly diseased trees appear near trees found infected in the previous year (Grzyb 1984, Jordovic 1975). This pattern seems to correspond to winged vectors introducing inoculum from outside of the orchard in the first few years, followed in later years by vector dissemination of the virus from infected trees within the orchard (Jordovic 1975).

Based on the pattern of spread to nearby trees, the estimated rate of spread averaged 10-15 m annually in the Soviet Union (Pomazkov and Abramova 1971). In Yugoslavia, the rate of infection over 10 years in 2 orchards ranged from 49 to 100 percent in orchards 100 m or less from a source of heavily infected trees, but dropped to less than 2 percent in orchards 500 m or more from the infection source. Besides distance from the source, some of the other factors affecting rate of spread are the number of infected trees (Jordovic 1968), climate, the tree cultivar, and the virus strain involved (European and Mediterranean Plant Protection Organization 1983). Spread in nurseries occurs rapidly because of such practices as planting material closely together and using undetected infected rootstocks or other infected material for propagation (Pemberton 1980).

Control

Using propagative material that is "virus-free" is an important means of exclusion. Planting orchards distant from infested orchards, planting cultivars tolerant to the virus, practicing vector control (Kegler et al. 1978), and roguing diseased trees are some of the other practices used to keep PPV under control.

Diseased trees should be eliminated before fall migrants return and disseminate PPV from these trees to healthy trees. Removing the diseased tree and surrounding symptomless trees was recommended (Kunze and Krczal 1971). All parts are best removed. Otherwise, burn leafy portions, and treat stump and suckers with systemic herbicide until no suckers emerge as suckers are usually infected. Replanting may then proceed (Adams 1978b, Pemberton 1980).

Roguing diseased trees in orchards with 10 percent infection or less kept PPV under control. In orchards with more than 10 percent infection, however, roguing was considered impractical and removing the entire orchard was recommended because that number of diseased trees meant too many trees to replace and too many latent infections that could serve as inoculum sources. That level also threatened adjacent orchards (Pemberton 1980).

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